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Recommendation of the Immunization Practices Advisory Committee (ACIP)

Rabies Prevention — United States, 1984

*These revised recommendations of the Immunization Practices Advisory Committee (ACIP) on rabies prevention update the previous recommendations (*MMWR *1980;29: 65-72,277-80) to reflect the current status of rabies and antirabies biologics in the United States. For assistance on problems or questions about rabies prophylaxis, call local or state health departments.**

INTRODUCTION

Although rabies rarely affects humans in the United States, every year, approximately 25,000 persons receive rabies prophylaxis. Appropriate managment of those who may have been exposed to rabies infection depends on the interpretation of the risk of infection and the efficacy and risk of prophylactic treatment. All available methods of systemic prophylactic treatment are complicated by instances of adverse reactions. These are rarely severe. Decisions on management must be made immediately; the longer treatment is postponed, the less likely it is to be effective.

Data on the efficacy of active and passive immunization after rabies exposure have come from both human and animal studies. Evidence from laboratory and field experience in many areas of the world indicates that postexposure prophylaxis combining local wound treatment, vaccine, and rabies immune globulin, is uniformly effective when appropriately used. However, rabies has occasionally developed in humans who had received postexposure antirabies prophylaxis with vaccine alone.

In the United States, rabies in humans has decreased from an average of 22 cases per year in 1946-1950 to zero to five cases per year since 1960. The number of rabies cases among domestic animals has decreased similarly. In 1946, more than 8,000 rabies cases were reported among dogs; 153 cases were reported in 1982. Thus, the likelihood of human exposure to rabies in domestic animals has decreased greatly, although bites by dogs and cats continue to be the principal reasons given for antirabies treatments.

The disease in wildlife—especially skunks, foxes, raccoons, and bats—has become more prevalent in recent years, accounting for approximately 85% of all reported cases of animal rabies every year since 1976. Wild animals now constitute the most important potential source of infection for both humans and domestic animals in the United States. Rabies among animals is present throughout the United States; only Hawaii remains consistently rabies-free.

Four of the six rabies fatalities in U.S. citizens occurring between 1980 and 1983 were related to exposure to rabid dogs outside the United States. In much of the world, including

*If these are unavailable, call the Division of Viral Diseases, Center for Infectious Diseases, CDC ([404] 329-3095 during working hours, or [404] 329-2888 nights, weekends, and holidays).

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most of Asia and all of Africa and Latin America, the dog remains the major source of human exposure.

RABIES IMMUNIZING PRODUCTS

There are two types of immunizing products: (1) vaccines that induce an active immune response, which requires about 7-10 days to develop but may persist for as long as a year or more, and (2) globulins that provide rapid passive immune protection, which persists for a short period of time, with a half-life of about 21 days. Both types of products should be used concurrently for rabies postexposure prophylaxis.

Vaccines for Use in the United States

Human diploid cell rabies vaccine (HDCV)†: HDCV is an inactivated virus vaccine prepared from fixed rabies virus grown in WI-38 or MRC-5 human diploid cell culture. The vaccine grown on WI-38 cells and developed in the United States is inactivated with tri-n-butyl phosphate and β-propiolactone (Wyeth Laboratories' WYVAC®), while that grown in MRC-5 cells and developed in Europe is inactivated with β-propiolactone (Merieux Institute's RABIES VACCINE®). Both vaccines are supplied as 1.0 ml, single-dose vials of lyophilized vaccine with accompanying diluent.

Globulins

Rabies Immune Globulin, Human (RIG): RIG (Cutter Laboratories' HYPERAB® and Merieux Institutes' IMOGAM®) is antirabies gamma globulin concentrated by cold ethanol fractionation from plasma of hyperimmunized human donors. Rabies neutralizing antibody content is standardized to contain 150 international units (IU) per ml. It is supplied in 2-ml (300 IU) and 10-ml (1,500 IU) vials for pediatric and adult use, respectively.

Antirabies Serum, Equine (ARS): ANTIRABIES SERUM® (Sclavo) is a refined, concentrated serum obtained from hyperimmunized horses. Neutralizing antibody content is standardized to contain 1,000 IU per vial. Volume is adjusted by the manufacturer on the basis of antibody potency in each lot. Currently, a 1,000-IU vial contains approximately 5 ml.

RATIONALE FOR CHOICE OF RABIES IMMUNIZING PRODUCTS

Both types of HDCV rabies vaccines are considered equally efficacious and safe when used as indicated on the labels. Only the Merieux Institute vaccine has been evaluated by the intradermal (ID) dose/route for preexposure immunization. No data are available on ID use with the Wyeth Laboratories vaccine. RIG is preferred over ARS, because the latter has a much higher risk of adverse reactions.

Vaccines

The effectiveness of rabies vaccines is measured by their ability to protect persons exposed to rabies and to induce antibodies to rabies virus. HDCV has been used concurrently with RIG or ARS to treat 45 persons bitten by rabid dogs or wolves in Iran, 31 persons bitten by a variety of rabid animals in Germany, and 511 persons bitten by a variety of rabid animals in the United States. In these studies, no person contracted rabies after receiving HDCV in combination with RIG.

All persons treated with RIG and five 1.0-ml intramuscular (IM) doses of HDCV and tested have developed a rabies antibody titer. The definition of a minimally acceptable antibody titer varies between laboratories and is influenced by the type of test conducted. CDC currently specifies a 1:5 titer by the rapid fluorescent-focus inhibition test (RFFIT) as acceptable. The World Health Organization (WHO) specifies a titer of 0.5 I.U.

Serious adverse reactions associated with rabies vaccines include systemic, anaphylactic, and neuroparalytic reactions. Serious adverse reactions occur at lower rates in the HDCV vaccine than with previously available types of rabies vaccine.

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†Official name: Rabies Vaccine. The duck embryo vaccine which was used from 1957-1982 is no longer available in the United States.

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Globulins

RIG and ARS are both effective; however, ARS causes serum sickness in over 40% of adult recipients. RIG rarely causes adverse reactions and should be the product of choice when available.

RATIONALE OF TREATMENT

Physicians must evaluate each possible rabies exposure. Local or state public health officials should be consulted if questions arise about the need for prophylaxis.

In the United States, the following factors should be considered before specific antirabies treatment is initiated:

Species of Biting Animal

Carnivorous wild animals (especially skunks, raccoons, foxes, coyotes, and bobcats) and bats are the animals most commonly infected with rabies and have caused most of the indigenous cases of human rabies in the United States since 1960. Unless an animal is tested and shown not to be rabid, postexposure prophylaxis should be initiated upon bite or nonbite exposure to the animals. (See definition in "Type of Exposure" below.) If treatment has been initiated and subsequent testing in a competent laboratory shows the exposing animal is not rabid, treatment can be discontinued.

The likelihood that a domestic dog or cat is infected with rabies varies from region to region; hence, the need for postexposure prophylaxis also varies.

Rodents (such as squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, and mice) and lagomorphs (including rabbits and hares) are rarely found to be infected with rabies and have not been known to cause human rabies in the United States. In these cases, the state or local health department should be consulted before a decision is made to initiate postexposure antirabies prophylaxis.

Circumstances of Biting Incident

An unprovoked attack is more likely than a provoked attack to indicate the animal is rabid. Bites inflicted on a person attempting to feed or handle an apparently healthy animal should generally be regarded as provoked.

Type of Exposure

Rabies is transmitted by introducing the virus into open cuts or wounds in skin or via mucous membranes. The likelihood of rabies infection varies with the nature and extent of exposure. Two categories of exposure should be considered.

Bite: Any penetration of the skin by teeth.

Nonbite: Scratches, abrasions, open wounds, or mucous membranes contaminated with saliva or other potentially infectious material, such as brain tissue, from a rabid animal. Casual contact, such as petting a rabid animal (without a bite or nonbite exposure as described above), does not constitute an exposure and is not an indication for prophylaxis. There have been two instances of airborne rabies acquired in laboratories and two probable airborne rabies cases acquired in a bat-infested cave in Texas.

The only documented cases of rabies from human-to-human transmission occurred in four patients in the United States and overseas who received corneas transplanted from persons who died of rabies undiagnosed at the time of death. Stringent guidelines for acceptance of donor corneas should reduce this risk.

Bite and nonbite exposures from humans with rabies theoretically could transmit rabies, although no cases of rabies acquired this way have been documented. Each potential exposure to human rabies should be carefully evaluated to minimize unnecessary rabies prophylaxis.

MANAGEMENT OF BITING ANIMALS

A healthy domestic dog or cat that bites a person should be confined and observed for 10 days and evaluated by a veterinarian at the first sign of illness during confinement or before

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release. Any illness in the animal should be reported immediately to the local health department. If signs suggestive of rabies develop, the animal should be humanely killed and its head removed and shipped, under refrigeration, for examination by a qualified laboratory designated by the local or state health department. Any stray or unwanted dog or cat that bites a person should be killed immediately and the head submitted, as described above, for rabies examination.

Signs of rabies in wild animals cannot be interpreted reliably; therefore, any wild animal that bites or scratches a person should be killed at once (without unnecessary damage to the head) and the brain submitted, as described above, for examination for evidence of rabies. If the brain is negative by fluorescent-antibody examination for rabies, the saliva can be assumed to contain no virus, and the bitten person need not be treated. If the biting animal is a particularly rare or valuable specimen and the risk of rabies small, consideration may be given to initiating postexposure treatment to the bitten person and delaying killing the animal for rabies testing.

POSTEXPOSURE PROPHYLAXIS

The essential components of rabies postexposure prophylaxis are local treatment of wounds and immunization, including administration, in most instances, of both globulin and vaccine (Tables 1 and 2).

Local Treatment of Wounds

Immediate and thorough washing of all bite wounds and scratches with *soap and water* is perhaps the most effective measure for preventing rabies. In experimental animals, simple local wound cleansing has been shown to reduce markedly the likelihood of rabies.

Tetanus prophylaxis and measures to control bacterial infection should be given as indicated.

Immunization

Postexposure antirabies immunization should always include administration of both antibody (preferably RIG) and vaccine, with one exception: persons who have been previously immunized with the recommended preexposure or postexposure regimens with HDCV or who have been immunized with other types of vaccines and have a history of documented adequate rabies antibody titer (See "RATIONALE FOR CHOICE OF RABIES IMMUNIZING PRODUCTS") should receive only vaccine. The combination of globulin and vaccine is recommended for both bite exposures and nonbite exposures (as described under "RATIONALE OF TREATMENT"), regardless of the interval between exposure and treatment. The sooner treatment is begun after exposure, the better. However, there have been instances in which the decision to begin treatment was made as late as 6 months or longer after the exposure due to delay in recognition that an exposure had occurred.

HDCV: HDCV is the only type of vaccine currently available in the United States and should be administered in conjunction with RIG at the beginning of postexposure therapy, as described below. In 1977, WHO established a recommendation for six IM doses of HDCV based on studies in Germany and Iran of a regimen of RIG or ARS and six doses of HDCV. When used in this way, the vaccine was safe and effective in protecting 76 persons bitten by proven rabid animals. The vaccine also induced an excellent antibody response in all recipients. Studies conducted by CDC in the United States have shown that a regimen of one dose of RIG and five doses of HDCV was safe and induced an excellent antibody response in all recipients. Of 511 persons bitten by proven rabid animals and so treated, none developed rabies.

Five 1-ml doses of HDCV should be given intramuscularly (for example, in the deltoid region). Other routes of administration, such as the ID route, have not been adequately evaluated for postexposure prophylaxis and should not be used. The first dose should be given as

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soon as possible after exposure; an additional dose should be given on days 3, 7, 14, and 28 after the first dose. (WHO currently recommends a sixth dose 90 days after the first dose.) Because the antibody response following the recommended vaccination regimen with HDCV has been so satisfactory, routine postvaccination serologic testing is not recommended. In unusual instances, as when the patient is known to be immunosuppressed, serologic testing is indicated. Contact state health department or CDC for recommendations.

RIG (or ARS if RIG is not available): RIG is administered only once, at the beginning of antirabies prophylaxis, to provide immediate antibodies until the patient responds to HDCV by active production of antibodies. If RIG was not given when vaccination was begun, it can be given up to the eighth day after the first dose of vaccine was given. From about the eighth day on, RIG is not indicated, since an antibody response to the vaccine is presumed to have occurred. The recommended dose of RIG is 20 IU/kg or approximately 9 IU/lb of body weight. (When ARS must be used, the recommended dose is 40 IU/kg, approximately 18 IU/lb or 1,000 IU/55 lb body weight.) If anatomically feasible, up to half the dose of RIG should be thoroughly infiltrated in the area around the wound, the rest should be administered intramuscularly. Because RIG may partially suppress active production of antibody, no more than the recommended dose of RIG should be given.

TABLE 1. Rabies postexposure prophylaxis guide – July 1984

The following recommendations are only a guide. In applying them, take into account the animal species involved, the circumstances of the bite or other exposure, the vaccination status of the animal, and presence of rabies in the region. Local or state public health officials should be consulted if questions arise about the need for rabies prophylaxis.

	Animal species	Condition of animal at time of attack	Treatment of exposed person*
DOMESTIC	Dog and cat	Healthy and available for 10 days of observation	None, unless animal develops rabies†
		Rabid or suspected rabid	RIG§ and HDCV
		Unknown (escaped)	Consult public health officials. If treatment is indicated, give RIG§ and HDCV
WILD	Skunk, bat, fox, coyote raccoon, bobcat, and other carnivores	Regard as rabid unless proven negative by laboratory tests¶	RIG§ and HDCV
OTHER	Livestock, rodents, and lagomorphs (rabbits and hares)	Consider individually. Local and state public health officials should be consulted on questions about the need for rabies prophylaxis. Bites of squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, mice, other rodents, rabbits, and hares almost never call for antirabies prophylaxis.	

**All bites and wounds should immediately be thoroughly cleansed with soap and water.* If antirabies treatment is indicated, both rabies immune globulin (RIG) and human diploid cell rabies vaccine (HDCV) should be given as soon as possible, *regardless* of the interval from exposure. Local reactions to vaccines are common and do not contraindicate continuing treatment. Discontinue vaccine if fluorescent-antibody tests of the animal are negative.

†During the usual holding period of 10 days, begin treatment with RIG and HDCV at first sign of rabies in a dog or cat that has bitten someone. The symptomatic animal should be killed immediately and tested.

§If RIG is not available, use antirabies serum, equine (ARS). Do not use more than the recommended dosage.

¶The animal should be killed and tested as soon as possible. Holding for observation is not recommended.

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TABLE 2. Rabies immunization — June 1984

I. PREEXPOSURE IMMUNIZATION. Preexposure immunization consists of three doses of HDCV, 1.0 ml, IM (i.e., deltoid area), one each on days 0, 7, and 28. (See text for details on use of 0.1 ml HDCV ID as an alternative dose/route.) Administration of routine booster doses of vaccine depends on exposure risk category as noted below. Preexposure immunization of immunosuppressed persons is not recommended.

	Criteria for Preexposure Immunization		
Risk category	**Nature of risk**	**Typical populations**	**Preexposure regimen**
Continuous	Virus present continuously, often in high concentrations. Aerosol, mucous membrane, bite, or nonbite exposure possible. Specific exposures may go unrecognized.	Rabies research lab workers.* Rabies biologics production workers.	Primary preexposure immunization course. Serology every 6 months. Booster immunization when antibody titer falls falls below acceptable level.*
Frequent	Exposure usually episodic, with source recognized, but exposure may also be unrecognized. Aerosol, mucous membrane, bite, or nonbite exposure.	Rabies diagnostic lab workers,* spelunkers, veterinarians, and animal control and wildlife workers in rabies epizootic areas.	Primary preexposure immunization course. Booster immunization or serology every 2 years.†
Infrequent (greater than population-at-large)	Exposure nearly always episodic with source recognized. Mucuous membrane, bite, or nonbite exposure.	Veterinarians and animal control and wildlife workers in areas of low rabies endemicity. Certain travelers to foreign rabies epizootic areas. Veterinary students.	Primary preexposure immunization course. No routine booster immunization or serology.
Rare (population-at-large)	Exposure always episodic, mucous membrane, or bite with source recognized.	U.S. population-at-large, including individuals in rabies-epizootic areas.	No preexposure immunization.

II. POSTEXPOSURE IMMUNIZATION. All postexposure treatment should begin with immediate thorough cleansing of all wounds with soap and water.

Persons not previously immunized:	RIG, 20 I.U./kg body weight, one half infiltrated at bite site (if possible), remainder IM; 5 doses of HDCV, 1.0 ml IM (i.e., deltoid area), one each on days 0, 3, 7, 14 and 28.
Persons previously immunized§:	Two doses of HDCV, 1.0 ml, IM (i.e., deltoid area), one each on days 0 and 3. RIG should not be administered.

*Judgment of relative risk and extra monitoring of immunization status of laboratory workers is the responsibility of the laboratory supervisor (see U.S. Department of Health and Human Service's *Biosafety in Microbiological and Biomedical Laboratories*, 1984).

†Preexposure booster immunization consists of one dose of HDCV, 1.0 ml/dose, IM (deltoid area). Acceptable antibody level is 1:5 titer (complete inhibition in RFFIT at 1:5 dilution). Boost if titer falls below 1:5.

§Preexposure immunization with HDCV; prior postexposure prophylaxis with HDCV; or persons previously immunized with any other type of rabies vaccine *and* a documented history of positive antibody response to the prior vaccination.

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TREATMENT OUTSIDE THE UNITED STATES

If postexposure is begun outside the United States with locally produced biologics, it may be desirable to provide additional treatment when the patient reaches the United States. State health departments should be contacted for specific advice in such cases.

PREEXPOSURE IMMUNIZATION

Preexposure immunization may be offered to persons in high-risk groups, such as veterinarians, animal handlers, certain laboratory workers, and persons spending time (e.g., 1 month or more) in foreign countries where rabies is a constant threat. Persons whose vocational or avocational pursuits bring them into contact with potentially rabid dogs, cats, foxes, skunks, bats, or other species at risk of having rabies should also be considered for preexposure prophylaxis.

Preexposure prophylaxis is given for several reasons. First, it may provide protection to persons with inapparent exposures to rabies. Second, it may protect persons whose postexposure therapy might be expected to be delayed. Finally, although it does not eliminate the need for additional therapy after a rabies exposure, it simplifies therapy by eliminating the need for globulin and decreasing the number of doses of vaccine needed. This is of particular importance for persons at high risk of being exposed in countries where the available rabies immunizing products may carry a higher risk of adverse reactions.

Preexposure immunization does not eliminate the need for prompt postexposure prophylaxis following an exposure; it only reduces the postexposure regimen.

Human Diploid Cell Rabies Vaccine

Three 1.0 ml injections of HDCV should be given intramuscularly (for example, in the deltoid area), one on each of days 0, 7, and 28. In a study in the United States, more than 1,000 persons received HDCV according to this regimen; antibody was demonstrated in the sera of all subjects when tested by the RFFIT. Other studies have produced comparable results. Because the antibody response following the recommended vaccination regimen with HDCV has been so satisfactory, routine postvaccination serology is not recommended.

Booster Doses of Vaccine

Persons who work with live rabies virus in research laboratories or vaccine production facilities and are at risk of inapparent exposure should have the rabies antibody titer of their serum determined every 6 months; booster doses of vaccine should be given, as needed, to maintain an adequate titer (See "RATIONALE FOR CHOICE OF RABIES IMMUNIZING PRODUCTS"). Other laboratory workers, such as those doing rabies diagnostic tests, spelunkers, and those veterinarians, animal control and wildlife officers in areas where animal rabies is epizootic should have boosters every 2 years or have their serum tested for rabies antibody every 2 years and, if the titer is inadequate, have a booster dose. Veterinarians and animal control and wildlife officers, if working in areas of low rabies endemicity, do not require routine booster doses of HDCV after completion of primary preexposure immunization (Table 2).

Postexposure Therapy of Previously Immunized Persons

When an immunized person who was vaccinated by the recommended regimen with HDCV or who had previously demonstrated rabies antibody is exposed to rabies, that person should receive two IM doses (1.0 ml each) of HDCV, one immediately and one 3 days later. RIG should not be given in these cases. If the immune status of a previously vaccinated person who did not receive the recommended HDCV regimen is not known, full primary postexposure antirabies treatment (RIG plus five doses of HDCV) may be necessary. In such cases, if antibody can be demonstrated in a serum sample collected before vaccine is given, treatment can be discontinued after at least two doses of HDCV.

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Intradermal Use of HDCV

HDCV produced by the Merieux Institute has been used for preexposure immunization in a regimen of three 0.1 ml doses given ID in the lateral aspect of the upper arm over the deltoid area, one dose each on days 0, 7, and 28. Experience gained with over 2,000 persons vaccinated in the United States by the ID route has shown that antibody was produced in all recipients, although the mean response was somewhat lower and may be of shorter duration than with comparable IM immunization. Antibody response in some groups vaccinated outside the United States has been found to be inadequate for reasons not yet determined.

Current data provide a sufficient basis to recommend the 0.1 ml ID dose/route as an alternative to the 1.0 ml IM dose/route for preexposure immunization in the United States. Postvaccination serology is not necessary following ID (or IM) immunization, except for persons suspected of being immunosuppressed. The manufacturer has not yet met the packaging and labeling requirements necessary to obtain approval by the U.S. Food and Drug Administration for the ID route. Since the 1.0-ml vial presently available is intended for IM use and contains no preservatives, the reconstituted vaccine must be used immediately. Data on ID immunization are not available for Wyeth Laboratories' vaccine, and it should not be used for ID vaccination.

ACCIDENTAL INOCULATION WITH MODIFIED LIVE RABIES VIRUS

Individuals may be accidentally exposed to attenuated rabies virus while administering modified live rabies virus (MLV) vaccines to animals. While there have been no reported human rabies cases resulting from exposure to needlesticks or sprays with licensed MLV vaccines, vaccine-induced rabies has been observed in animals given MLV vaccines. Absolute assurance of a lack of risk for humans, therefore, cannot be given. The best evidence for a low risk, however, is the absence of recognized cases of vaccine-associated disease in humans despite frequent accidental exposures.

Currently available MLV animal vaccines are made with one of two attenuated strains of rabies virus: high egg passage (HEP) Flury strain or Street Alabama Dufferin (SAD) strain. The HEP Flury and SAD virus strains have been used in animal vaccines for over 10 years without evidence of associated disease in humans; therefore, postexposure treatment is not recommended following exposure to these types of vaccine by needlesticks or sprays.

Because the data are insufficient to assess the true risk associated with any of the MLV vaccines, preexposure immunization, and periodic boosters are recommended for all persons dealing with potentially rabid animals or frequently handling animal rabies vaccines.

ADVERSE REACTIONS

Human Diploid Cell Rabies Vaccine

Reactions after vaccination with HDCV are less common than with previously available vaccines. In a study using five doses of HDCV, local reactions, such as pain, erythema, and swelling or itching at the injection site, were reported in about 25% of recipients of HDCV, and mild systemic reactions, such as headache, nausea, abdominal pain, muscle aches, and dizziness were reported in about 20% of recipients. Two cases of neurologic illness resembling Guillain-Barré syndrome that resolved without sequelae in 12 weeks, and a focal subacute central nervous system disorder temporally associated with HDCV vaccine, have been reported.

Recently, a significant increase has been noted in "immune complex-like" reactions in persons receiving booster doses of HDCV. The illness, characterized by onset 2-21 days postbooster, presents with a generalized urticaria and may also include arthralgia, arthritis, angioedema, nausea, vomiting, fever, and malaise. In no cases were the illnesses life-threatening. Preliminary data suggest this "immune complex-like" illness may occur in up to 6% of persons receiving booster vaccines and much less frequently in persons receiving primary immuniza-

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tion. Additional experience with this vaccine is needed to define more clearly the risk of these adverse reactions.

Vaccines in Other Countries

Many developing countries use inactivated nerve tissue vaccines (NTV) or inactivated suckling mouse brain vaccine (SMBV). NTV is reported to provoke neuroparalytic reactions at a rate of about 1/2,000 vaccinees; the rate for SMBV is about 1/8,000.

Rabies Immune Globulin, Human

Local pain and low-grade fever may follow receipt of RIG. Although not reported specifically for RIG, angioneurotic edema, nephrotic syndrome, and anaphylaxis have been reported after injection of immune serum globulin (ISG). These reactions occur so rarely that the causal relationship between ISG and these reactions is not clear.

Antirabies Serum, Equine

ARS produces serum sickness in at least 40% of adult recipients; reaction rates for children are lower. Anaphylactic reactions may occur. When RIG is not available, and ARS must be used, the patient should be tested for sensitivity to equine serum. (See package circular for details.)

Because adverse reactions are associated more frequently with ARS than with RIG, and ARS might sensitize recipients to equine protein, ARS should be used only when RIG cannot be obtained.

Management of Adverse Reactions

Once initiated, rabies prophylaxis should not be interrupted or discontinued because of local or mild systemic adverse reactions to rabies vaccine. Usually such reactions can be successfully managed with anti-inflammatory and antipyretic agents (aspirin, for example).

When a person with a history of hypersensitivity must be given rabies vaccines, antihistamines may be given; epinephrine should be readily available to counteract anaphylactic reactions, and the person should be carefully observed immediately after immunization.

Serious systemic anaphylactic or neuroparalytic reactions occurring during the administration of rabies vaccines pose a serious dilemma for the attending physician. A patient's risk of developing rabies must be carefully considered before deciding to discontinue vaccination. Moreover, the use of corticosteroids to treat life-threatening neuroparalytic reactions carries the risk of inhibiting the development of active immunity to rabies. It is especially important in these cases that the serum of the patient be tested for rabies antibodies. Advice and assistance on the management of serious adverse reactions in persons receiving rabies vaccines may be sought from the state health department or CDC.

All serious systemic neuroparalytic or anaphylactic reactions to a rabies vaccine should be immediately reported to the state health department or the Division of Viral Diseases, Center for Infectious Diseases, CDC ([404] 329-3095 during working hours, or [404] 329-2888 at other times).

PRECAUTIONS AND CONTRAINDICATIONS

Immunosuppression

Corticosteroids, other immunosupressive agents, and immunosuppressive illnesses can interfere with the development of active immunity and predispose the patient to developing rabies. Immunosuppressive agents should not be administered during postexposure therapy, unless essential for the treatment of other conditions. When rabies postexposure prophylaxis is administered to persons receiving steroids or other immunosuppressive therapy, it is especially important that serum be tested for rabies antibody to ensure that an adequate response has developed.

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Pregnancy

Because of the potential consequences of inadequately treated rabies exposure and limited data that indicate that fetal abnormalities have not been associated with rabies vaccination, pregnancy is not considered a contraindication to postexpsoure prophylaxis. If there is substantial risk of exposure to rabies, preexposure prophylaxis may also be indicated during pregnancy.

Allergies

Persons with histories of hypersensitivity should be given rabies vaccines with caution. When a patient with a history suggesting hypersensitivity to HDCV must be given that vaccine, antihistamines can be given; epinephrine should be readily available to counteract anaphylactic reactions, and the person should be carefully observed.

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TABLE I. Summary—cases specified notifiable diseases, United States

Disease	28th Week Ending			Cumulative, 18th Week Ending		
	July 14, 1984	July 16, 1983	Median 1979-1983	July 14, 1984	July 16, 1983	Median 1979-1983
Acquired Immunodeficiency Syndrome (AIDS)	79	N	N	2,130	N	N
Aseptic meningitis	114	248	193	2,345	2,863	2,515
Encephalitis: Primary (arthroped-borne & unspec.)	16	37	37	445	526	485
Post-infectious	5	1	1	60	54	54
Gonorrhea: Civilian	14,381	17,832	19,276	425,249	470,894	509,293
Military	544	388	375	10,979	12,762	14,562
Hepatitis: Type A	265	303	498	10,976	11,247	13,582
Type B	401	430	399	13,141	12,449	10,670
Non A, Non B	55	69	N	1,950	1,821	N
Unspecified	69	99	163	3,118	3,799	5,351
Legionellosis	12	8	N	300	362	N
Leprosy	3	8	6	121	139	107
Malaria	25	34	34	438	394	539
Measles: Total*	12	2	33	1,892	1,111	2,323
Indigenous	11	2	N	1,713	936	N
Imported	1	-	N	179	175	N
Meningococcal infections: Total	49	28	42	1,717	1,767	1,767
Civilian	49	28	42	1,713	1,751	1,751
Military	-	-	-	4	16	12
Mumps	15	23	44	1,959	2,136	3,964
Pertussis	28	74	27	1,042	1,058	628
Rubella (German measles)	11	12	31	449	697	1,784
Syphilis (Primary & Secondary): Civilian	400	520	495	14,703	17,148	15,929
Military	3	5	5	178	228	196
Toxic Shock syndrome	5	6	N	227	254	N
Tuberculosis	412	513	513	11,314	12,196	14,119
Tularemia	11	10	9	103	130	108
Typhoid fever	3	15	13	160	191	219
Typhus fever, tick-borne (RMSF)	26	62	54	347	470	479
Rabies, animal	56	107	107	2,669	3,509	3,509

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1984		Cum. 1984
Anthrax	1	Plague	11
Botulism: Foodborne	6	Poliomyelitis: Total	2
Infant (Mont. 1, Calif. 1)	49	Paralytic	2
Other (Calif. 1)	4	Psittacosis (Calif. 3)	46
Brucellosis (Minn. 1)	51	Rabies, human	-
Cholera	-	Tetanus (Mo. 1)	25
Congenital rubella syndrome	3	Trichinosis (Mass. 1, Ohio 1, Alaska 2)	44
Diphtheria	-	Typhus fever, flea-borne (endemic, murine) (Calif. 1)	10
Leptospirosis (Ohio 1)	10		

*One of the 12 reported cases for this week was imported from a foreign country or can be directly traceable to a known internationally imported case within two generations.

TABLE III. Cases of specified notifiable diseases, United States, weeks ending July 14, 1984 and July 16, 1983 (28th Week)

Reporting Area	AIDS	Aseptic Menin-gitis	Encephalitis		Gonorrhea (Civilian)		Hepatitis (Viral), by type				Legionel-losis	Leprosy
			Primary	Post-in-fectious			A	B	NA,NB	Unspeci-fied		
	Cum. 1984	1984	Cum. 1984	Cum. 1984	Cum. 1984	Cum. 1983	1984	1984	1984	1984	1984	Cum. 1984
UNITED STATES	2,130	114	445	80	425,249	470,694	285	401	55	89	12	121
NEW ENGLAND	71	8	29	1	12,030	11,677	5	16	2	14	1	5
Maine	-	-	-	-	492	600	-	1	-	-	-	-
N.H.	1	3	4	-	336	360	-	5	1	-	-	-
Vt.	-	1	2	-	200	223	-	-	-	-	-	-
Mass.	37	2	15	-	4,598	5,050	4	6	1	12	1	4
R.I.	4	-	-	-	832	638	-	-	-	-	-	1
Conn.	29	2	8	1	5,572	4,806	1	4	-	2	-	-
MID ATLANTIC	963	9	55	6	58,665	59,886	21	50	4	2	-	24
Upstate N.Y.	83	4	20	5	8,880	9,327	5	7	-	-	-	2
N.Y. City	695	3	3	-	24,627	24,493	9	29	-	2	-	22
N.J.	138	-	15	-	9,947	11,149	-	-	-	-	-	-
Pa.	47	2	17	1	15,211	14,917	7	14	4	-	-	-
E.N. CENTRAL	104	12	96	15	56,677	67,448	14	30	4	4	2	6
Ohio	14	4	34	7	14,788	18,061	5	8	-	-	1	2
Ind.	16	4	20	-	6,734	7,016	4	6	1	3	1	-
Ill.	54	-	14	6	12,185	18,941	2	7	1	1	-	2
Mich.	14	4	23	-	16,302	17,708	3	9	2	-	-	2
Wis.	6	-	5	2	6,668	5,722	-	-	-	-	-	-
W.N. CENTRAL	21	5	16	-	20,380	21,995	6	17	2	-	-	1
Minn.	5	-	6	-	3,018	3,099	-	2	-	-	-	-
Iowa	1	-	7	-	2,288	2,415	-	3	-	-	-	1
Mo.	10	4	1	-	9,829	10,755	4	7	2	-	-	-
N. Dak.	-	-	-	-	194	228	-	-	-	-	-	-
S. Dak.	-	-	-	-	520	611	1	2	-	-	-	-
Nebr.	2	1	1	-	1,321	1,361	-	3	-	-	-	-
Kans.	3	-	1	-	3,210	3,526	1	-	-	-	-	-
S. ATLANTIC	301	33	80	14	108,574	120,812	20	114	8	6	5	5
Del.	4	-	1	-	1,959	2,159	-	1	1	-	-	-
Md.	19	3	19	-	12,094	15,507	-	16	-	-	-	-
D.C.	42	-	-	-	7,891	8,182	-	6	-	-	-	1
Va.	17	5	19	5	10,297	10,400	1	9	-	-	2	3
W. Va.	4	2	5	-	1,297	1,284	-	2	-	-	-	-
N.C.	6	6	16	7	17,114	17,689	-	19	1	2	2	-
S.C.	6	-	2	-	10,689	11,479	1	16	1	1	-	-
Ga.	28	3	2	1	20,864	25,255	4	18	-	-	1	-
Fla.	175	14	16	1	26,369	28,857	14	27	5	3	-	1
E.S. CENTRAL	14	15	22	6	36,885	39,680	9	45	6	7	-	-
Ky.	7	-	3	-	4,474	4,598	5	2	-	2	-	-
Tenn.	3	1	6	1	15,326	16,255	2	10	2	1	-	-
Ala.	3	12	12	5	11,797	12,309	2	31	4	4	-	-
Miss.	1	2	1	-	5,288	6,518	-	2	-	-	-	-
W.S. CENTRAL	115	2	32	4	57,931	66,598	32	19	3	19	1	7
Ark.	-	1	-	2	4,919	5,059	1	-	-	3	-	-
La.	18	-	4	-	13,230	11,928	20	14	2	12	-	-
Okla.	4	1	10	1	6,343	7,827	6	5	1	1	1	-
Tex.	93	-	18	1	33,439	41,784	5	-	-	3	-	7
MOUNTAIN	32	7	17	7	13,752	14,594	28	29	3	7	2	7
Mont.	-	-	-	-	578	635	-	-	-	-	1	-
Idaho	-	-	-	-	663	662	-	4	1	-	-	-
Wyo.	1	-	-	-	402	384	-	-	-	-	-	-
Colo.	19	5	7	-	3,979	4,110	6	6	-	-	-	-
N. Mex.	-	-	-	-	1,548	1,771	1	2	1	-	-	-
Ariz.	6	2	4	3	3,753	4,073	13	10	1	3	-	5
Utah	3	-	6	4	667	721	4	-	-	-	1	1
Nev.	3	-	-	-	2,162	2,238	4	7	-	4	-	1
PACIFIC	509	23	98	7	60,355	68,004	130	81	23	10	1	66
Wash.	25	5	3	-	4,131	5,197	4	4	6	-	-	3
Oreg.	3	-	-	-	3,589	3,533	23	4	2	-	-	1
Calif.	476	15	93	7	50,126	56,176	102	72	15	10	1	47
Alaska	-	1	-	-	1,501	1,685	-	-	-	-	-	-
Hawaii	5	2	2	-	1,008	1,413	1	1	-	-	-	15
Guam	-	U	-	-	95	94	U	U	U	U	U	-
P.R.	33	2	-	1	1,835	1,524	13	13	-	12	-	1
V.I.	-	-	-	-	228	154	-	-	-	-	-	-
Pac. Trust Terr.	-	U	-	-	-	-	U	U	U	U	U	-

N: Not notifiable U: Unavailable

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending July 14, 1984 and July 16, 1983 (29th Week)

Reporting Area	Malaria	Measles (Rubeola)					Menin-gococcal Infections	Mumps		Pertussis			Rubella		
		Indigenous		Imported *		Total									
	Cum. 1984	1984	Cum. 1984	1984	Cum. 1984	Cum. 1983	Cum. 1984	1984	Cum. 1984	1984	Cum. 1984	Cum. 1983	1984	Cum. 1984	Cum. 1983
UNITED STATES	438	11	1,713	1	175	1,111	1,717	15	1,969	25	1,042	1,058	11	449	897
NEW ENGLAND	28	1	98	-	9	15	102	-	60	2	20	38	-	28	11
Maine	-	-	-	-	-	-	1	-	16	-	-	4	-	1	-
N.H.	-	-	33	-	3	3	6	-	13	-	4	6	-	-	3
Vt.	2	1	3	-	3	-	23	-	3	2	14	7	-	-	3
Mass.	15	-	52	-	-	4	38	-	14	-	1	17	-	27	5
R.I.	4	-	-	-	-	-	9	-	5	-	1	4	-	-	-
Conn.	7	-	10	-	3	8	27	-	9	-	-	-	-	-	-
MID ATLANTIC	70	4	94	1	22	77	285	1	228	3	92	244	4	145	123
Upstate N.Y.	19	1	18	-	7	6	102	1	52	3	55	78	-	97	20
N.Y. City	16	3	72	1†	9	41	49	-	12	-	3	36	4	38	86
N.J.	21	-	4	-	2	27	67	-	126	-	5	15	-	11	3
Pa.	14	-	-	-	4	3	77	-	38	-	29	115	-	1	14
E.N. CENTRAL	33	2	585	-	67	613	270	3	814	19	288	258	-	67	109
Ohio	7	-	2	-	5	78	94	3	417	2	51	75	-	2	1
Ind.	-	-	2	-	1	393	36	-	40	16	196	20	-	2	22
Ill.	10	-	159	-	1	136	53	-	155	-	15	110	-	38	45
Mich.	6	2	392	-	54	5	52	-	152	1	13	12	-	18	15
Wis.	10	-	10	-	6	1	35	-	50	-	12	41	-	7	26
W.N. CENTRAL	12	-	2	-	3	1	110	1	80	1	80	63	1	28	30
Minn.	2	-	-	-	3	1	21	-	3	-	9	22	-	2	6
Iowa	1	-	-	-	-	-	18	-	17	1	4	5	1	1	-
Mo.	6	-	2	-	-	-	32	-	7	-	12	11	-	-	-
N. Dak.	1	-	-	-	-	-	1	-	1	-	-	1	-	3	-
S. Dak.	-	-	-	-	-	-	7	-	-	-	5	3	-	-	-
Nebr.	1	-	-	-	-	-	9	-	3	-	2	-	-	-	-
Kans.	1	-	-	-	-	-	22	1	49	-	48	21	-	22	24
S. ATLANTIC	78	-	10	-	17	177	359	2	137	1	75	152	-	20	84
Del.	4	-	-	-	-	-	3	-	2	-	2	2	-	-	-
Md.	19	-	4	-	5	5	29	-	27	-	4	25	-	1	1
D.C.	1	-	-	-	5	-	5	-	-	-	-	-	-	-	-
Va.	19	-	1	-	1	22	41	1	13	-	9	42	-	-	1
W. Va.	1	-	-	-	-	-	5	-	27	-	7	5	-	-	-
N.C.	5	-	-	-	-	-	51	-	15	-	17	17	-	-	9
S.C.	1	-	-	-	-	4	34	-	2	-	1	11	-	-	1
Ga.	6	-	-	-	-	8	71	-	17	-	5	31	-	2	11
Fla.	22	-	5	-	6	138	120	1	34	1	30	19	-	17	61
E.S. CENTRAL	3	-	1	-	2	6	100	-	37	-	6	11	-	7	10
Ky.	-	-	1	-	-	1	38	-	8	-	1	3	-	3	9
Tenn.	-	-	-	-	2	-	24	-	12	-	2	3	-	-	-
Ala.	3	-	-	-	-	5	28	-	5	-	-	3	-	1	1
Miss.	-	-	-	-	-	-	12	-	12	-	3	2	-	3	-
W.S. CENTRAL	34	-	382	-	22	70	184	-	105	-	231	143	-	13	88
Ark.	-	-	-	-	-	10	27	-	5	-	11	13	-	3	-
La.	5	-	-	-	-	25	35	-	-	-	3	2	-	-	9
Okla.	5	-	-	-	7	1	23	N	N	-	208	104	-	-	-
Tex.	24	-	382	-	15	34	99	-	100	-	11	24	-	10	79
MOUNTAIN	16	-	91	-	10	3	58	2	193	1	74	104	1	13	27
Mont.	1	-	-	-	-	-	1	-	4	-	17	1	-	-	3
Idaho	2	-	-	-	-	-	6	-	8	-	3	3	-	1	8
Wyo.	-	-	-	-	-	-	2	-	1	-	3	4	-	2	2
Colo.	1	-	-	-	-	2	20	-	13	1	26	70	-	2	-
N. Mex.	1	-	68	-	8	-	7	N	N	-	5	8	-	1	-
Ariz.	8	-	-	-	-	1	14	2	161	-	13	9	-	-	6
Utah	3	-	23	-	2	-	5	-	5	-	5	9	-	6	7
Nev.	-	-	-	-	-	-	3	-	1	-	2	-	1	1	1
PACIFIC	164	4	490	-	27	149	249	6	305	1	178	45	5	128	215
Wash.	5	-	107	-	-	4	36	-	32	1	33	8	-	1	8
Oreg.	8	-	-	-	-	7	37	N	N	-	11	6	-	-	13
Calif.	148	-	244	-	24	137	168	5	254	-	86	31	5	123	194
Alaska	-	-	-	-	-	-	7	1	5	-	-	-	-	1	-
Hawaii	3	4	139	-	3	1	1	-	14	-	69	-	-	3	-
Guam	1	U	83	U	2	2	1	U	5	U	-	-	U	2	-
P.R.	3	-	-	-	-	81	3	1	82	-	-	8	-	6	3
V.I.	-	-	-	-	-	5	-	-	3	-	-	-	-	-	1
Pac. Trust Terr.	-	U	-	U	-	-	-	U	-	U	-	-	U	-	-

*For measles only, imported cases includes both out-of-state and international importations.

N: Not notifiable U: Unavailable †International §Out-of-state

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending July 14, 1984 and July 16, 1983 (28th Week)

Reporting Area	Syphilis (Civilian) (Primary & Secondary)		Toxic-shock Syndrome	Tuberculosis		Tula-remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1984	Cum. 1983	1984	Cum. 1984	Cum. 1983	Cum. 1984	Cum. 1984	Cum. 1984	Cum. 1984
UNITED STATES	14,703	17,148	5	11,314	12,196	103	160	347	2,869
NEW ENGLAND	294	383	-	316	352	2	7	1	21
Maine	3	10	-	17	20	-	-	-	10
N.H.	7	16	-	22	25	-	-	-	4
Vt.	1	1	-	7	4	-	-	-	-
Mass.	172	236	-	166	179	2	5	1	5
R.I.	11	13	-	25	28	-	-	-	-
Conn.	100	107	-	80	96	-	2	-	2
MID ATLANTIC	2,007	2,171	-	2,078	2,199	-	23	4	171
Upstate N.Y.	134	175	-	356	342	-	9	3	16
N.Y. City	1,255	1,280	-	826	892	-	6	1	-
N.J.	369	419	-	459	462	-	4	-	4
Pa.	249	297	-	435	503	-	4	-	151
E.N. CENTRAL	636	942	1	1,484	1,571	1	22	16	117
Ohio	131	247	1	285	251	-	4	12	11
Ind.	74	73	-	168	146	-	2	2	13
Ill.	177	465	-	620	689	1	8	-	48
Mich.	210	113	-	317	404	-	2	2	13
Wis.	44	44	-	94	81	-	6	-	32
W.N. CENTRAL	223	210	-	322	396	28	6	26	446
Minn.	67	88	-	58	80	-	2	-	45
Iowa	10	9	-	34	37	-	-	1	88
Mo.	109	74	-	156	207	18	3	4	37
N. Dak.	5	1	-	8	5	-	-	-	87
S. Dak.	2	9	-	11	28	10	-	3	116
Nebr.	11	11	-	16	11	-	-	2	31
Kans.	19	18	-	39	28	-	1	16	42
S. ATLANTIC	4,404	4,490	1	2,382	2,436	4	19	160	768
Del.	16	19	-	31	20	-	-	-	4
Md.	288	286	-	263	189	-	-	13	438
D.C.	174	191	-	88	94	-	6	-	-
Va.	227	321	-	234	242	-	4	26	133
W. Va.	10	15	-	76	81	-	-	5	23
N.C.	437	417	1	349	334	1	1	57	10
S.C.	406	278	-	282	231	-	1	42	28
Ga.	751	835	-	322	453	3	1	16	86
Fla.	2,117	2,128	-	717	792	-	6	1	48
E.S. CENTRAL	978	1,169	-	1,041	1,121	2	5	34	136
Ky.	57	67	-	238	272	-	2	5	35
Tenn.	276	326	-	339	332	2	2	18	58
Ala.	313	483	-	314	294	-	1	6	45
Miss.	332	293	-	150	223	-	-	5	-
W.S. CENTRAL	3,535	4,503	-	1,290	1,451	48	9	99	569
Ark.	89	108	-	137	160	30	-	16	61
La.	849	951	-	166	254	3	1	1	23
Okla.	121	121	-	127	126	13	2	61	68
Tex.	2,676	3,323	-	861	911	-	6	19	417
MOUNTAIN	336	375	-	289	340	15	10	5	118
Mont.	2	5	-	14	34	-	1	5	62
Idaho	14	6	-	18	18	4	-	-	-
Wyo.	4	7	-	-	8	-	-	-	-
Colo.	78	81	-	25	33	5	2	-	21
N. Mex.	44	115	-	56	72	1	3	-	9
Ariz.	131	90	-	133	136	2	3	-	21
Utah	11	13	-	27	23	2	-	-	-
Nev.	52	58	-	16	16	1	1	-	5
PACIFIC	2,290	2,905	3	2,134	2,330	5	59	2	323
Wash.	72	105	-	107	112	-	1	-	1
Oreg.	70	60	1	88	100	2	1	1	1
Calif.	2,104	2,696	2	1,789	1,945	3	53	-	315
Alaska	3	7	-	33	33	-	1	1	6
Hawaii	41	37	-	117	140	-	3	-	-
Guam	-	-	U	5	4	-	-	-	-
P.R.	459	598	-	217	263	-	3	-	34
V.I.	8	10	-	2	1	-	3	-	-
Pac. Trust Terr.	-	-	U	-	-	-	-	-	-

U: Unavailable

TABLE IV. Deaths in 121 U.S. cities,* week ending July 14, 1984 (28th Week Ending)

Reporting Area	All Causes, By Age (Years)						P&I** Total
	All Ages	≥65	45-64	25-44	1-24	<1	
NEW ENGLAND	677	484	125	37	14	17	41
Boston, Mass.	176	121	37	10	5	3	16
Bridgeport, Conn.	38	22	12	3	-	1	-
Cambridge, Mass.	29	25	3	1	-	-	2
Fall River, Mass.	37	30	5	1	1	-	-
Hartford, Conn.	73	45	16	7	3	2	1
Lowell, Mass.	33	24	5	3	1	-	4
Lynn, Mass.	15	13	2	-	-	-	1
New Bedford, Mass.	32	26	4	2	-	-	1
New Haven, Conn.	58	38	10	5	1	4	2
Providence, R.I.	67	45	7	1	-	4	7
Somerville, Mass.	4	4	-	-	-	-	-
Springfield, Mass.	36	26	6	1	1	2	1
Waterbury, Conn.	33	23	7	1	2	-	2
Worcester, Mass.	56	42	11	2	-	1	4
MID. ATLANTIC	2,569	1,664	566	212	72	55	95
Albany, N.Y.	52	34	9	3	2	4	-
Allentown, Pa.	33	18	14	-	1	-	-
Buffalo, N.Y.	118	71	34	6	3	2	10
Camden, N.J.	40	22	11	5	-	2	-
Elizabeth, N.J.	26	19	4	3	-	-	-
Erie, Pa.†	31	19	7	2	2	1	-
Jersey City, N.J.	46	32	8	3	1	2	-
N.Y. City, N.Y.	1,365	860	294	144	43	24	47
Newark, N.J.	58	33	13	5	4	3	4
Paterson, N.J.	37	21	7	4	2	3	2
Philadelphia, Pa.†	294	191	66	24	5	8	12
Pittsburgh, Pa.†	49	30	17	-	1	1	2
Reading, Pa.	30	20	8	-	2	-	-
Rochester, N.Y.	128	99	20	5	2	2	6
Schenectady, N.Y.	32	26	6	-	-	-	1
Scranton, Pa.†	28	20	7	1	-	-	-
Syracuse, N.Y.	118	83	25	4	3	3	6
Trenton, N.J.	27	16	7	3	1	-	3
Utica, N.Y.	24	20	4	-	-	-	1
Yonkers, N.Y.	35	30	5	-	-	-	1
E.N. CENTRAL	2,247	1,396	531	161	77	82	72
Akron, Ohio	57	31	15	8	3	-	-
Canton, Ohio	40	30	6	2	1	1	-
Chicago, Ill.	547	306	153	42	19	28	10
Cincinnati, Ohio	115	80	19	9	2	5	3
Cleveland, Ohio	182	100	42	10	4	6	2
Columbus, Ohio	129	73	35	11	4	6	6
Dayton, Ohio	105	60	27	4	9	5	7
Detroit, Mich.	252	149	64	20	11	8	6
Evansville, Ind.	47	37	5	3	2	-	1
Fort Wayne, Ind.	60	33	13	5	3	6	4
Gary, Ind.	15	8	3	3	1	-	1
Grand Rapids, Mich.	45	35	10	-	-	-	1
Indianapolis, Ind.	183	107	48	17	7	6	2
Madison, Wis.	40	28	7	3	1	1	6
Milwaukee, Wis.	131	91	21	11	2	6	3
Peoria, Ill.	50	35	11	2	2	-	7
Rockford, Ill.	36	28	3	3	2	-	1
South Bend, Ind.	58	41	15	2	-	-	5
Toledo, Ohio	108	75	22	5	3	3	5
Youngstown, Ohio	67	50	14	1	1	1	2
W.N. CENTRAL	808	545	165	45	21	32	28
Des Moines, Iowa	91	66	13	7	-	5	7
Duluth, Minn.	39	29	9	1	-	-	1
Kansas City, Kans.	33	19	8	3	2	1	-
Kansas City, Mo.	103	59	29	9	1	5	2
Lincoln, Nebr.	35	28	5	1	1	-	-
Minneapolis, Minn.	78	55	15	3	2	3	4
Omaha, Nebr.	96	64	17	7	3	5	5
St. Louis, Mo.	181	115	34	13	9	10	-
St. Paul, Minn.	67	54	10	1	1	1	3
Wichita, Kans.	85	56	25	-	2	2	6
S. ATLANTIC	1,298	773	303	128	56	38	32
Atlanta, Ga.	142	88	27	17	6	4	-
Baltimore, Md.	249	154	61	17	11	6	5
Charlotte, N.C.	84	53	18	7	2	4	1
Jacksonville, Fla.	79	50	17	4	4	4	3
Miami, Fla.	173	108	44	13	8	2	1
Norfolk, Va.	47	26	13	3	4	1	-
Richmond, Va.	85	44	31	6	2	2	-
Savannah, Ga.	33	18	7	5	3	-	3
St. Petersburg, Fla.	115	94	16	3	1	1	7
Tampa, Fla.	67	40	18	6	1	2	6
Washington, D.C.	181	73	40	45	12	11	3
Wilmington, Del.	43	27	11	2	2	1	3
E.S. CENTRAL	708	444	181	39	18	25	36
Birmingham, Ala.	107	67	26	8	1	5	2
Chattanooga, Tenn.	60	34	22	2	1	1	1
Knoxville, Tenn.	82	48	23	5	3	3	7
Louisville, Ky.	87	53	23	4	2	5	3
Memphis, Tenn.	141	94	27	10	6	3	5
Mobile, Ala.	73	49	16	3	2	3	5
Montgomery, Ala.	51	35	13	3	-	-	2
Nashville, Tenn.	107	64	31	4	3	5	11
W.S. CENTRAL	1,365	755	330	137	76	67	38
Austin, Tex.	44	28	6	3	5	2	4
Baton Rouge, La.	45	28	7	4	4	2	-
Corpus Christi, Tex.	36	18	11	4	3	-	-
Dallas, Tex.	209	110	59	25	8	7	4
El Paso, Tex.	69	37	17	4	6	5	4
Fort Worth, Tex.	100	64	21	7	4	4	3
Houston, Tex.	427	208	118	60	20	21	5
Little Rock, Ark.	59	30	17	3	4	5	4
New Orleans, La.	92	50	18	14	7	3	-
San Antonio, Tex.	177	112	35	9	8	13	13
Shreveport, La.	37	22	9	2	2	2	-
Tulsa, Okla.	70	48	12	2	5	3	1
MOUNTAIN	651	391	147	57	35	21	32
Albuquerque, N.Mex.	78	49	12	12	2	3	6
Colo. Springs, Colo.	39	26	7	2	3	1	5
Denver, Colo.	132	72	34	11	11	4	6
Las Vegas, Nev.	72	39	19	7	6	1	3
Ogden, Utah	32	20	5	4	1	2	1
Phoenix, Ariz.	147	86	43	7	4	7	2
Pueblo, Colo.	25	17	4	2	2	-	2
Salt Lake City, Utah	31	19	9	2	-	1	-
Tucson, Ariz.	95	63	14	10	6	2	7
PACIFIC	2,045	1,340	439	147	61	53	90
Berkeley, Calif.	29	23	6	-	-	-	-
Fresno, Calif.	75	47	11	3	6	8	3
Glendale, Calif.	28	21	4	2	1	-	2
Honolulu, Hawaii	83	49	23	8	1	2	5
Long Beach, Calif.	80	55	23	2	-	-	3
Los Angeles, Calif.	565	331	145	55	17	12	11
Oakland, Calif.	71	45	19	4	2	1	10
Pasadena, Calif.	42	32	7	3	-	-	2
Portland, Oreg.	121	80	32	4	1	4	6
Sacramento, Calif.	151	101	28	11	5	6	16
San Diego, Calif.	150	116	21	7	3	3	11
San Francisco, Calif.	152	104	25	19	2	2	3
San Jose, Calif.	205	139	36	16	9	5	10
Seattle, Wash.	146	101	26	8	5	6	2
Spokane, Wash.	59	37	14	1	4	3	2
Tacoma, Wash.	88	59	19	4	5	1	4
TOTAL	12,368††	7,792	2,787	963	430	390	464

* Mortality data in this table are voluntarily reported from 121 cities in the United States, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included.

** Pneumonia and influenza

† Because of changes in reporting methods in these 4 Pennsylvania cities, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

†† Total includes unknown ages.

TABLE V. Years of potential life lost, deaths, and death rates, by cause of death, and estimated number of physician contacts, by principal diagnosis, United States

Cause of morbidity or mortality (Ninth Revision ICD, 1975)	Years of potential life lost before age 65 by persons dying in 1982*†	Estimated mortality February 1984		Estimated number of physician contacts February 1984*¶
		Number*§	Annual Rate/100,000*§	
ALL CAUSES (TOTAL)	9,429,000	165,960	920.6	104,800,000
Accidents and adverse effects (E800-E949)	2,367,000	8,270	34.8	5,400,000
Malignant neoplasms (140-208)	1,809,000	35,390	196.3	1,500,000
Diseases of heart (390-398, 402, 404-429)	1,566,000	62,680	347.7	5,200,000
Suicides, homicides (E950-E978)	1,314,000	3,520	19.5	—
Cerebrovascular diseases (430-438)	256,000	13,340	74.0	600,000
Chronic liver disease and cirrhosis (571)	252,000	2,340	13.0	100,000
Pneumonia and influenza (480-487)	118,000	5,700	31.6	3,300,000
Chronic obstructive pulmonary diseases and allied conditions (490-496)	114,000	5,770	32.0	2,400,000
Diabetes mellitus (250)	106,000	2,850	15.8	2,200,000
Prenatal care*				2,900,000
Infant mortality*††		3,700	12.9 /1,000 live births	

*For details of calculation, see footnotes for Table V, *MMWR* 1984;33:2.

†Years of potential life lost for persons between 1 year and 65 years old at the time of death are derived from the number of deaths in each age category as reported by the National Center for Health Statistics, *Monthly Vital Statistics Report* (MVSR), Vol. 31, No. 13, October 5, 1983.

§National Center for Health Statistics, *Monthly Vital Statistics Report* (MVSR), Vol. 33, No. 3, June 21, 1984, pp. 8-9.

¶IMS America *National Disease and Therapeutic Index* (NDTI), Monthly Report, February 1984, Section III.

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Epidemiologic Notes and Reports

Chromosomally Mediated Resistant *Neisseria gonorrhoeae* — United States

During 1983-1984, an increasing number of cases of β-lactamase negative, penicillin-resistant *Neisseria gonorrhoeae* were reported to CDC. Unlike penicillinase-producing *N. gonorrhoeae* (PPNG), which have plasmid-mediated resistance to penicillin, these β-lactamase negative, resistant gonococci have chromosomally mediated resistance based on available data.

The first reported outbreak of chromosomally mediated (β-lactamase negative) resistant *N. gonorrhoeae* (CMRNG) in the United States occurred in Durham County, North Carolina (*1*). Since this outbreak, in which more than 200 cases were eventually detected, 16 other states have reported cases with resistant gonococci. Of these, Tennessee, New Mexico, and Oregon have reported more sustained outbreaks.

Cases in these outbreaks were detected either by routine screening of all gonococcal isolates (New Mexico) or screening of primary treatment failure isolates (Tennessee, Oregon) for susceptibility to penicillin at the local or state levels. Screening was performed by disk agar diffusion or by growth on penicillin-containing media. Gonococcal isolates that grew on media containing 1.6 μg/ml of penicillin or produced a zone of inhibition less than 26 mm, with a 10 μg penicillin disk, were submitted to CDC for confirmation of resistance. Minimum inhibitory concentrations by the agar dilution susceptibility test were determined for antimicrobials that included penicillin, ampicillin, tetracycline, cefotaxime, cefuroxime, cefoxitin, spectinomycin, and trimethoprim/sulfamethoxazole. Isolates resistant to penicillin and ampicillin were equally resistant to tetracycline by agar dilution susceptibility testing.

Of all CMRNG isolates submitted to CDC for agar dilution susceptibility testing during 1983-1984, 11.0% were susceptible to less than 2 μg/ml of penicillin; none were susceptible to less than 2 μg/ml of tetracycline; and only 47.0% were susceptible to less than 0.5 μg/ml trimethoprim and 9.5 μg/ml sulfamethoxazole (trimethoprim/sulfamethoxazole). All isolates were susceptible to spectinomycin, cefoxitin, cefuroxime, and cefotaxime. Immunologic characterization demonstrated that all CMRNG isolates were serogroup IIb (the majority of the same serovariant) based on serotyping by experimental monoclonal antibodies to major outer membrane protein (*2*). Of the 18 New Mexico cases, two distinctly different serovariants were detected within serogroup IIb.

Neisseria gonorrhoeae — *Continued*

Clinical and epidemiologic information were obtained for patients whose isolates were tested. Excluding North Carolina, of the 16 other reporting states, over half of the CMRNG cases were from Tennessee, New Mexico, and Oregon.

Tennessee: All the 14 Tennessee patients were heterosexuals, and two patients could be linked to interstate travel to Virginia or North Carolina. Strains from the Tennessee cases were immunologically similar and had similar antimicrobial susceptibility patterns consistent with continued endemic transmission within the state.

New Mexico: Of the 18 CMRNG patients from New Mexico, seven were heterosexual (three males, four females), and 11 were homosexual males. All heterosexual patients and seven homosexual patients were infected with gonococcal strains immunologically identical, with similar antimicrobial susceptibility patterns. Strains from these cases were more resistant to penicillin than strains from the other four homosexual patients. Heterosexual CMRNG patients could not be linked to homosexual CMRNG patients by sexual history or naming of sexual contacts. All homosexual patients were clustered within Albuquerque; heterosexual patients were more widely distributed throughout the state. Based on immunologic studies of the gonoccoci recovered from these individuals and examination of temporal and geographic variables for heterosexuals versus homosexuals, at least two separate outbreaks with no demonstrable common source occurred in New Mexico. No evidence for interstate or foreign transmission into New Mexico could be identified for any of the cases.

Oregon: Of the eight cases reported from Oregon, all occurred among homosexual males. Gonococcal strains from these individuals shared identical immunologic and antimicrobial susceptibility patterns. No epidemiologic evidence for interstate or foreign transmission could be documented for any of these cases, suggesting only endemic transmission within the homosexual community in Oregon. No additional cases have been reported from Oregon since March 1984.

Reported by M Kimberly, DrPh, State Laboratory Director, W DeVault, CE Chapman, MD, G Conrad, Venereal Disease Control, RH Hutcheson, Jr, MD, State Epidemiologist, Tennessee State Dept of Health; JM Mann, MD, L Nims, Scientific Laboratory, A Chowning, E Montes, Venereal Disease Control, HF Hull, MD, State Epidemiologist, Health Svcs Div, New Mexico Dept of Health and Environment, L Foster, MD, D Harger, H Horton, Venereal Disease Control, C Schade, MD, JA Googins, MD, State Epidemiologist, State Health Div, Oregon Dept of Human Resources; Sexually Transmitted Diseases Laboratory Program, Center for Infectious Diseases, Div of Sexually Transmitted Diseases, Center for Prevention Svcs, Div of Field Svcs, Epidemiology Program Office, CDC.

Editorial Note: Seventeen states, including North Carolina, have reported cases of CMRNG to CDC since 1983. The majority of these cases were detected as primary therapeutic failures to the penicillins or tetracyclines. Gonococcal strains from the majority of U.S. outbreaks and cases have generally been immunologically similar (serogroup IIb) with similar antimicrobial susceptibilities.

Based on epidemiologic data, foreign importation has been infrequently documented for these CMRNG strains in the United States (*3*). In contrast, foreign importation contributes to the largest proportion of PPNG in the United States, although domestic transmission became more important after 1976 (*4*).

Cases of CMRNG may be detected by screening for penicillin resistance at the local or state levels to guide appropriate therapy and permit rapid follow-up of cases. Screening by disk agar diffusion or with penicillin-containing media will identify chromosomally mediated resistance to penicillin. Disk susceptibility testing to tetracycline and trimethoprim/sulfamethoxazole should be performed only by standardized procedures using appropriate controls (*5,6*). Inconsistent results to these two antimicrobials may be seen with disk susceptibility testing (*5,6*).

Based on agar dilution susceptibility testing, infections caused by CMRNG should clinically respond to therapy with recommended dosages of spectinomycin, cefoxitin, cefotaxime, or ce-

Neisseria gonorrhoeae — *Continued*

furoxime. CDC treatment guidelines for PPNG infections provide the recommended schedules for these antimicrobials and emphasize the importance of the immediate use of spectinomycin as primary therapy for gonorrhea cases when treatment failures are suspected (*7*).

Since 1975, gonorrhea has generally declined in the United States (*8*). PPNG increased dramatically between 1976 and 1982 but decreased in 1983 (*8*). Unfortunately, cases of CMRNG have been reported with increasing frequency since the North Carolina outbreak. Because the extent and prevalence of CMRNG infections are not yet fully understood, screening of all β-lactamase negative (nonpenicillinase-producing) primary treatment failure gonococcal isolates for penicillin susceptibility (*1*) is encouraged at the local and state levels to improve surveillance and guide appropriate therapy. Screening at the community level should be most cost-effective, since the majority of these CMRNG strains are equally resistant to tetracycline, thereby preventing unnecessary and usually ineffective retreatment with a tetracycline. Because of high secondary treatment failure rates with tetracycline, tetracycline should not be used as the drug of choice for either PPNG or CMRNG infections that have failed primary therapy with penicillin or ampicillin. Spectinomycin, cefoxitin, or cefotaxime should be used to treat CMRNG infections at dosages recommended for PPNG (*7*).

More active surveillance for these CMRNG infections will be required to determine their accurate prevalence, and support control activities.

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Fatalities from Occupational Heat Exposure

Presented below are two of several fatalities from occupational heat stroke reported to the National Institute for Occupational Safety and Health (NIOSH) since 1977.

Indiana: In July 1980, a 24-year-old white male, who was employed at a surface coal mine, collapsed and later died after performing heavy labor in a hot environment. The worker, 5 feet 9 inches tall and weighing about 200 pounds, had been employed at the mine for 1½ weeks. On the day of the reported incident, he was assigned to load 40-pound bags of explosives into vertically drilled holes in preparation for blasting the material overlying the coal seam. He began work at 6:00 a.m., and at 3:40 p.m., informed a co-worker that he did not feel well. He walked about 50 yards to a shady area and collapsed. The outdoor dry bulb temperature was 39.4 C (103 F).

The worker was moved to a nearby hospital where his rectal temperature registered 42.2 C (108 F). By the time he was transferred to the intensive care unit (ICU), his temperature exceeded 43.3 C (110 F). He was treated with an ice pack and intravenous fluids but died at 6:30 p.m. The autopsy report listed systemic hyperthermia with extreme generalized dilation of capillaries (cardiovascular shock) and cerebral edema as the immediate causes of death.

Occupational Heat Exposure — Continued

Wisconsin: In September 1981, a 39-year-old black male, 5 feet 7 inches tall and weighing 165 pounds, was employed as a furnace attendant at an aluminum foundry. He had worked at the foundry for 2 weeks and was responsible for turning on and attending a furnace used to melt aluminum. On the afternoon of the reported incident, he had pressed the wrong button and accidentally spilled molten aluminum on the floor. He spent about 15 minutes removing the spill and wore a silver reflective suit for protection against the radiant heat emanating from the metal. The outdoor dry bulb temperature was 28.3 C (83 F), and the worksite temperature was about 29.4 C (84 F); the estimated temperature of the molten aluminum in the furnace was 982.2 C (1,800 F).

After removing the spilled material, the worker described the accident to his supervisor and, still wearing the suit, left the workplace without explanation. He was discovered 15 minutes later having seizures in the foundry parking lot. Paramedics transported him to a hospital at 5:40 p.m.; on arrival, his body temperature was 41.7 C (107 F). Medication controlled the seizures, but he remained comatose. He was treated with rubbing alcohol and an ice pack, and at 7:00 p.m., when his body temperature was 35.6 C (96 F), he was placed on a hyperthermic machine in the ICU. He began bleeding from the rectum at 9:30 p.m., and fresh, frozen plasma was administered. The bleeding apparently stopped but then recurred with hematuria. He died the next day at 9:30 a.m. in cardiac arrest. The autopsy report listed the causes of death as hyperthermia, disseminated intravascular coagulation, and coronary arteriosclerosis.

The worker had a history of treatment for alcoholism and reportedly had been drinking heavily in the days before his death; however, at the time of hospitalization, he had no alcohol in his blood. Four days before the heatstroke, he had severely lacerated his toes in a lawnmower accident and was treated with antibiotics and tetanus toxoid.

Reported by Div of Respiratory Disease Studies, Div of Biomedical and Behavioral Science, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Illness and death from environmental heat are important public health problems (*1*). This is especially true in the occupational setting when workers performing physical labor outdoors are exposed to higher-than-normal ambient temperatures and when such temperatures have an additive effect on heat generated by the jobs themselves. The fatalities reported here illustrate, in both outdoor and indoor settings, the circumstances that may lead to heatstroke and, subsequently, to death.

Occupational heat-related conditions include heat cramps, heat exhaustion, dehydration, and skin disorders. In addition, the risk of unintentional injuries increases substantially with exposure to heat stress (*2*). An estimated six million workers in the United States may be exposed to occupational heat stress. Estimates of deaths and illnesses associated with occupational heat exposures are difficult to obtain, because worksite conditions and occupation are usually not listed on hospital records or death certificates; moreover, heatstroke may not be recognized as the primary cause of illness or death. However, for 1973-1976, annual reports from the California Department of Health Services alone show seven fatalities among 1,128 acute occupational heat-related illnesses (*3*). About 10%-15% of these patients required hospitalization, and an additional 40% were absent from work for varying periods after their illnesses; the remainder returned to work after medical treatment.

The health status of a worker is important in determining the response to heat exposure (*4*). Certain preexisting conditions can render a person more susceptible to heatstroke; these include obesity, drug abuse, alcoholism, acute or chronic illnesses, fatigue, poor physical condition, overeating, use of anticholinergic and certain psychotropic drugs, lack of sleep, and lack of acclimatization (*5*). The first worker described here was moderately obese and in poor physical condition; the second had a history of treatment for alcoholism and may have been affected by the wound and the medication he received 4 days before his death.

Occupational Heat Exposure – Continued

In 1969, an international panel of scientists convened by the World Health Organization recommended keeping a worker's deep body temperature at or below 38 C (100.4 F) to prevent heat illnesses (*6*). In response to this, NIOSH developed in 1972 a Criteria Document for Occupational Exposure to Hot Environments, which recommended the following preventive measures (*7*): (1) acclimatizing new workers and workers returning from vacation or absence because of illness; (2) implementing a work/rest regimen matched to the severity of the workers' heat exposure. (The Threshold Limit Value for Heat Stress adopted by the American Conference of Governmental Industrial Hygienists can be used as a guide to establish a suitable work/rest regimen [*8*]); (3) scheduling hot operations for the coolest part of the day; (4) making drinking water and salt readily available to replace the water and salt lost by sweating; (5) making protective clothing available to workers, as appropriate; (6) reducing environmental heat by engineering controls; (7) monitoring environmental heat at the job site; (8) performing pre-employment and periodic medical examinations to define those at increased risk; and (9) instructing workers and supervisors about preventive measures and early recognition of the symptoms of heat-related disorders.

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Current Trends

Tuberculosis — United States, 1983

In 1983, 23,846 cases of tuberculosis were reported to CDC, for a rate of 10.2 cases per 100,000 population. Compared with 1982, this represents a 6.6% decrease in the number of cases reported and a decline of 7.3% in the rate.

Rates for the 50 states ranged from 23.1/100,000 in Hawaii to 1.3/100,000 in North Dakota (Table 3). The rate increased in 13 states, remained unchanged in one, and decreased in 36.

The rate among persons living in 56 cities with populations of 250,000 or more was 21.2/100,000—more than twice the national rate (Table 4). Urban rates ranged from 58.4/100,000 in Miami, Florida, to 2.5/100,000 in Toledo, Ohio. Eight cities had rates at least three times the national rate: Miami, Florida; Newark, New Jersey; Atlanta, Georgia; San Francisco, California; Tampa, Florida; Honolulu, Hawaii; Washington, D.C.; and Oakland, California.

Tuberculosis — Continued

TABLE 3. Tuberculosis cases and rates — United States, 1983 and 1982

State	Tuberculosis cases		Case rate		Rank according to rate		Population
	1983	1982	1983	1982	1983	1982	July 1, 1983
United States	23,846	25,520	10.2	11.0	*	*	233,981,000
Alabama	522	631	13.2	16.0	11	7	3,959,000
Alaska	98	96	20.5	21.9	2	2	479,000
Arizona	264	300	8.9	10.5	22	21	2,963,000
Arkansas	414	412	17.8	18.0	3	3	2,328,000
California	3,469	3,606	13.8	14.6	7	9	25,174,000
Colorado	108	113	3.4	3.7	42	41	3,139,000
Connecticut	194	155	6.2	4.9	33	36	3,138,000
Delaware	65	55	10.7	9.1	17	23	606,000
District of Columbia†	202	228	32.4	36.1	*	*	623,000
Florida	1,457	1,467	13.6	14.1	9	11	10,680,000
Georgia	808	830	14.1	14.7	5	8	5,732,000
Hawaii	236	252	23.1	25.4	1	1	1,023,000
Idaho	35	31	3.5	3.2	40	45	989,000
Illinois	1,380	1,653	12.0	14.4	15	10	11,486,000
Indiana	411	399	7.5	7.3	27	31	5,479,000
Iowa	65	73	2.2	2.5	47	47	2,905,000
Kansas	76	92	3.1	3.8	44	39	2,425,000
Kentucky	523	605	14.1	16.5	6	4	3,714,000
Louisiana	439	471	9.9	10.8	19	19	4,438,000
Maine	39	57	3.4	5.0	43	35	1,146,000
Maryland	409	540	9.5	12.7	20	16	4,304,000
Massachusetts	389	503	6.7	8.7	30	26	5,767,000
Michigan	790	864	8.7	9.5	23	22	9,069,000
Minnesota	165	157	4.0	3.8	38	40	4,144,000
Mississippi	414	333	16.0	13.1	4	14	2,587,000
Missouri	399	390	8.0	7.9	26	28	4,970,000
Montana	47	37	5.8	4.6	35	37	817,000
Nebraska	25	32	1.6	2.0	49	49	1,597,000
Nevada	52	67	5.8	7.6	34	29	891,000
New Hampshire	38	33	4.0	3.5	39	43	959,000
New Jersey	809	804	10.8	10.8	16	18	7,468,000
New Mexico	116	122	8.3	9.0	24	25	1,399,000
New York	2,309	2,268	13.1	12.8	12	15	17,667,000
North Carolina	780	806	12.8	13.4	13	12	6,082,000
North Dakota	9	16	1.3	2.4	50	48	680,000
Ohio	519	621	4.8	5.8	37	33	10,746,000
Oklahoma	331	335	10.0	10.5	18	20	3,298,000
Oregon	182	194	6.8	7.3	28	30	2,662,000
Pennsylvania	972	1,090	8.2	9.1	25	24	11,895,000
Rhode Island	60	34	6.3	3.5	32	42	955,000
South Carolina	443	513	13.6	16.0	10	6	3,264,000
South Dakota	46	36	6.6	5.2	31	34	700,000
Tennessee	645	747	13.8	16.1	8	5	4,685,000
Texas	1,965	2,045	12.5	13.4	14	13	15,724,000
Utah	46	51	2.8	3.3	45	44	1,619,000
Vermont	11	13	2.1	2.5	48	46	525,000
Virginia	520	672	9.4	12.2	21	17	5,550,000
Washington	239	301	5.6	7.1	36	32	4,300,000
West Virginia	133	162	6.8	8.3	29	27	1,965,000
Wisconsin	164	208	3.5	4.4	41	38	4,751,000
Wyoming	14	10	2.7	2.0	46	50	514,000
American Samoa§	7	4	20.4	12.1	*	*	34,298
Guam§	48	49	45.4	46.3	*	*	105,821
Northern Mariana Is.§	74	75	441.0	443.8	*	*	16,780
Puerto Rico§	452	473	13.9	14.8	*	*	3,261,000
Trust Terr. Pacific Is.§	188	209	160.7	178.6	*	*	116,973
U.S. Virgin Is.§	2	0	2.0	0.0	*	*	101,500

*Not ranked.

†District of Columbia is not ranked with the States but is included in totals.

§Not included in totals.

Tuberculosis — Continued

TABLE 4. Tuberculosis cases and rates: cities with populations of 250,000 or more — United States, 1983 and 1982

State	Tuberculosis cases		Case rate		Rank according to rate		Population estimates
	1983	1982	1983	1982	1983	1982	1983
Albuquerque, N.M.	25	30	7.0	8.6	53	46	357,600
Atlanta, Ga.	191	*	43.8	*	3	*	436,000
Austin, Tex.	33	40	8.8	11.0	51	39	375,500
Baltimore, Md.	148	221	19.7	29.0	18	10	750,000
Birmingham, Ala.	74	74	26.2	25.8	11	13	282,500
Boston, Mass.	137	150	24.3	26.8	15	11	563,000
Buffalo, N.Y.	50	42	14.8	12.4	32	35	338,100
Charlotte, N.C.	45	59	13.7	18.7	38	25	328,400
Chicago, Ill.	871	1,069	29.0	35.6	10	6	3,005,100
Cincinnati, Ohio	60	78	15.6	20.2	27	20	385,500
Cleveland, Ohio	88	125	15.3	21.8	28	17	573,800
Columbus, Ohio	43	51	11.8	9.0	43	44	364,900
Dallas, Tex.	215	190	22.6	20.4	17	19	949,600
Denver, Colo.	49	56	9.8	11.2	48	38	500,600
Detroit, Mich.	286	312	25.1	25.9	12	12	1,138,700
El Paso, Tex.	66	76	14.0	16.7	36	29	471,600
Ft. Worth, Tex.	76	76	18.7	19.1	19	24	406,300
Honolulu, Hawaii	135	131	35.3	35.3	6	7	382,200
Houston, Tex.	517	648	29.4	38.1	9	4	1,760,000
Indianapolis, Ind.	102	92	14.4	13.0	33	34	706,800
Jacksonville, Fla.	82	91	14.8	16.5	31	30	554,400
Kansas City, Mo.	43	42	9.6	9.4	49	43	448,200
Long Beach, Cal.	60	95	16.1	25.6	26	14	373,100
Los Angeles, Cal.	769	684	25.0	22.5	13	16	3,071,100
Louisville, Ky.	74	*	24.8	*	14	*	298,700
Memphis, Tenn.	89	*	13.6	*	40	*	655,600
Miami, Fla.	225	269	58.4	61.4	1	1	385,100
Milwaukee, Wisc.	65	63	10.5	10.0	45	42	618,200
Minneapolis, Minn.	40	31	11.0	8.5	44	47	364,700
Nashville, Tenn.	75	*	16.3	*	25	*	459,900
Newark, N.J.	159	145	49.9	44.4	2	2	318,800
New Orleans, La.	99	121	17.5	21.5	22	18	567,200
New York, N.Y.	1,651	1,594	23.3	22.5	16	15	7,086,100
Norfolk, Va.	37	52	13.9	19.4	37	23	266,900
Oakland, Cal.	110	35	31.7	10.1	8	41	347,300
Oklahoma City, Okla.	55	46	13.4	11.3	41	37	409,700
Omaha, Nebr.	12	12	3.8	3.8	55	52	312,900
Philadelphia, Pa.	297	335	17.8	19.8	21	21	1,665,400
Phoenix, Ariz.	87	89	10.3	10.8	46	40	841,200
Pittsburgh, Pa.	65	75	15.3	17.7	29	28	424,000
Portland, Ore.	67	66	18.4	17.9	20	27	365,000
Sacramento, Cal.	42	94	14.4	32.9	34	8	292,600
St. Louis, Mo.	57	59	13.6	13.4	39	33	419,800
St. Paul, Minn.	27	22	10.1	8.2	47	48	267,300
San Antonio, Tex.	136	133	16.4	16.3	24	31	830,400
San Diego, Cal.	131	138	14.2	15.4	35	32	925,000
San Francisco, Cal.	303	299	42.9	43.2	4	3	705,700
San Jose, Cal.	101	118	15.0	17.9	30	26	671,800
Seattle, Wash.	85	97	17.4	19.7	23	22	489,700
Tampa, Fla.	100	83	36.5	30.7	5	9	274,300
Toledo, Ohio	9	26	2.5	7.3	56	50	354,600
Tucson, Ariz.	47	41	13.1	11.6	42	36	359,900
Tulsa, Okla.	34	32	9.3	8.8	50	45	365,900
Virginia Beach, Va.	19	17	6.7	6.2	54	51	282,600
Washington, D.C.	202	228	32.4	36.1	7	5	623,000
Wichita, Kans.	20	23	7.1	8.2	52	49	279,800
Total—56 Cities	8,685	8,775	21.2	22.3	†	†	41,052,100
San Juan, P.R.	79	80	17.8	18.4	†	†	443,600

*Not available, because in 1982, the reporting area included city-county data.

†Not ranked.

Tuberculosis – Continued

In 1983, 1,360 tuberculosis cases were reported among children under 15 years of age, including 818 cases among children less than 5 years of age; in 1982, there were 1,349 and 789 such cases, respectively.

Final tuberculosis mortality data for 1981 show 1,937 deaths. Compared with the final totals of 2,007 and 1,978 deaths in 1979 and 1980 and the 1982 provisional estimate of 1,980 deaths by the National Center for Health Statistics, there was essentially no change in tuberculosis mortality over the 4-year period 1979-1982.

Reported by Div of Tuberculosis Control, Center for Prevention Svcs, CDC.

Editorial Note: From 1968 through 1978, the average annual decrease in tuberculosis cases in the United States was 5.6%. From 1978 through 1981, when there was a large influx of Southeast Asian refugees, the average annual decline was only 1.4%. A 6.8% decrease in the number of cases in 1982 and the 6.6% decrease in 1983 indicate the previous downward trend has resumed.

Three factors may have contributed to the decreased number of tuberculosis cases reported in 1983: (1) There was an increase in the number of states using the new individual case reporting system, which requires more accurate verification of cases before they are counted; (2) the number of refugees arriving in the United States with tuberculosis declined; and (3) the number of indigenous tuberculosis cases may have actually declined.

Despite the decline in reported cases in 1983, tuberculosis persists as a public health problem. Transmission of infection continues, as evidenced by the continued occurrence and lack of decline of disease in young children. Tuberculosis mortality has not declined; moreover, in 1980, tuberculosis was the leading cause of death among 38 notifiable diseases for which mortality data were reported (*1*). The number of tuberculosis deaths that year exceeded the combined total of deaths for the other 37 notifiable diseases. It is estimated that more than 10 million persons in this country are infected with tubercle bacilli. They have a lifelong risk of developing disease, which can be minimized by giving preventive treatment. Additional cases will occur in new residents of this country who come from areas of the world where tuberculosis infection rates are much higher than in the United States. Unless otherwise contraindicated, these persons should receive a course of preventive therapy (*2*).

State and local health departments are responsible for ensuring the control of tuberculosis in the community. It is estimated that 40,000 persons on health department registers are currently under treatment or medical supervision for tuberculosis and that each year, approximately 200,000 persons exposed to new cases must be examined. Many of these persons are placed on preventive treatment. Tuberculosis control has been complicated by the global emergence of organisms resistant to antituberculous drugs (*3*). Community outbreaks continue to occur in the United States (*4,5*).

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The data in this report are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the succeeding Friday.

The editor welcomes accounts of interesting cases, outbreaks, environmental hazards, or other public health problems of current interest to health officials. Such reports and any other matters pertaining to editorial or other textual considerations should be addressed to: ATTN: Editor, *Morbidity and Mortality Weekly Report*, Centers for Disease Control, Atlanta, Georgia 30333.

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